The first surgical correction of a human patent ductus arteriosus (PDA) was performed in 1938. Less than 2 decades later, Dr. Willis Potts was the first to perform surgical ligation of a PDA in a dog. Today, PDA is recognized as one of the most common congenital heart defects in dogs, with an incidence ranging from 25% to 30% of cases. Female toy-breed dogs are overrepresented in this condition. A characteristic history and clinical signs, along with a classic “machinery” murmur, typically lead to procedures such as chest radiography and echocardiography for confirmation. Depending on the size and duration of the defect, diagnostics may reveal left ventricular heart enlargement, mitral regurgitation, and overcirculation of the pulmonary vasculature. Most dogs with PDA develop congestive heart failure (CHF) by 1 year of age if ligation or occlusion is not performed. Immature dogs (younger than 1 year) appear to be the best candidates for surgery. Some dogs are born with or may develop suprasystemic pulmonary hypertension that can result in reversal of flow through the PDA (i.e., blood flows from the pulmonary artery into the aorta). This is referred to as a right-to-left PDA. Occlusion is contraindicated in patients with right-to-left shunting of blood through the PDA. Symptomatic medical therapy is the only recommended course of treatment in these cases.

**Abstract:** Patent ductus arteriosus (PDA) is the most common congenital heart disease in dogs. It is due to the failure of the ductus arteriosus muscle to constrict, leaving a passageway for blood flow and resulting in eventual left-sided heart disease and/or generalized heart failure. It is hereditary in several breeds. The typical left-to-right PDA is amenable to minimally invasive procedures or open surgery. The ideal surgical candidate for PDA occlusion is immature and lightweight, with minimal heart changes. There is a wide variety of surgical techniques involving different methods of dissection and suture passage. Intraoperative hemorrhage during dissection is the most serious potential complication and can be life-threatening. Minimally invasive techniques such as thorascopic ligation and intravascular coiling have been claimed to have lower morbidity and mortality than open techniques. Once the PDA is occluded, most patients have remodeling of the myocardial tissues, resulting in an excellent long-term prognosis. Late complications such as residual flow and recanalization are rare but may be clinically significant.

**Physiology and Pathophysiology**

The ductus arteriosus (DA), also referred to as the arterial duct, arterial canal, and ductus Botalli, forms the sixth aortic arch. The DA extends from the bifurcation of the main pulmonary artery to the ventral aspect of the descending aorta between the left subclavian artery and the intercostal arteries. It normally comprises 98% smooth muscle, with subadventitial elastic fibers and loose collagen intermingled within the adventitia. In the fetus, the DA shunts blood away from the nonfunctional lungs back to the systemic circulation. At birth, the neonate’s lungs expand. This allows dilation of the pulmonary arterioles and a profound reduction in the pulmonary vascular resistance to approximately 20% of the systemic resistance. Pulmonary vascular resistance is further diminished by thinning of the smooth muscle within the pulmonary arterioles. During this time, an increase in systemic oxygen tension stimulates the smooth muscle within the DA to constrict in a process called apobiosis. Diminished circulating prostaglandin also plays a role in DA closure. In utero, circulating prostaglandin levels are high due to placental production and minimal pulmonary metab-
Transverse histologic section of a normally constricted ductus (D) in a 3-day-old mixed-breed dog. The ductus muscle is circumferentially uniform. The aorta (A) and pulmonary artery (P) have thicker elastic fibers.

Transverse histologic section of a PDA (D) and adjacent aorta (A) and pulmonary artery (P) in an 11-day-old dog with a grade 5 PDA. The ductus muscle (DM) is asymmetrically constricted. The portion adjacent to the aorta is not constricted and has a thicker elastic segment.

At birth, the placenta no longer serves as a source of prostaglandins, and prostaglandin metabolism by the lungs increases. With the inhibiting influence of prostaglandins dwindling, the DA is able to close. The closed DA predominantly comprises uniform, circumferential smooth muscle cells and very little elastic tissue (FIGURE 1). Physiologic closure of the DA occurs immediately after birth; anatomic closure follows within a 48 hours to 1 month. By 1 month of age, the muscle cells degenerate via cytolysis and the DA becomes a nonpatent elastic structure referred to as the ligamentum arteriosum.

In dogs with PDA, the DA fails to contract due to asymmetric distribution of the muscular component (FIGURE 2). Overall, the proportion of noncontractile elastic tissue to smooth muscle mass in the DA wall is greater in these dogs, and this interspersed elastic tissue prevents the smooth muscle cells from completely closing the vessel. The result is a PDA that is generally funnel-shaped, with the narrowest portion adjacent to the pulmonary artery. Typically, a fibrous ridge of incomplete ductus muscle within the PDA lumen incompletely narrows the orifice. A system has been developed for the histologic grading of PDA based on the amount of abnormal elastic tissue present (FIGURE 3). The severity of the grade increases with the proportion of defective genome inherited from affected parents. In one study, dogs with large PDAs that had a reversal of flow typically had the smallest amount of smooth muscle within the PDA wall (i.e., a grade 6 PDA) and conversely had the greatest amount of elastic tissue in the wall.

The PDA courses within the wall of the aorta before emptying into the aortic lumen, forming an aortoductal aneurysm. Generally, the size of the aortoductal aneurysm varies inversely with the length of the ductus: the shorter the surgical segment, the larger the aneurysm and the more difficult surgical occlusion may be. Several authors argue that the aneurysm is the result of turbulent blood flow created as the blood is impeded by this intraaortic shelf. Buchanan suggested that the aortic aneurysm of a PDA develops because the ductal component within the aorta renders that portion of the aortic wall less rigid.

A PDA allows blood to flow from the systemic circulation (aorta) into the pulmonary circulation (i.e., left-to-right shunting). The blood flow through the PDA is continuous throughout the cardiac cycle as long as the blood pressure of the systemic circulation remains higher than the blood pressure of the pulmonary circulation. The greatest pressure difference between the two systems exists at the end of systole, when aortic pressure is 120 mm Hg and pulmonic artery pressure is 20 mm Hg. The resulting gradient of 100 mm Hg across the PDA is equivalent to a blood flow of 5 m/sec. This results in volume overload in the pulmonary system, as demonstrated by dilation of the pulmonary arteries and veins, left atrium, and left ventricle. Because this volume overload is chronic, the left side of the heart undergoes eccentric hypertrophy. Eventually, this left-sided over-
load leads to left-sided CHF that can become generalized as the right side of the heart continues to pump blood through a fibrotically stenosed pulmonary vasculature to a decom-pensating left heart. In a patient with a PDA, this can occur as early as 1 week of age or may develop many years later; however, 70% of dogs with PDA develop clinical signs of CHF before 12 months of age. The timeline for the development of CHF appears to depend on the diameter of the PDA. A dog with a small PDA may not display clinical signs of CHF until later in life. There is a report of a 15-year-old cocker spaniel with a small, incidental PDA identified at necropsy. In the worst cases, a large PDA may allow bidirectional or right-to-left shunting within the first month of life.

Heritability

Although several early epidemiologic studies suggested a genetic component to the occurrence of PDA, in 1971, Patterson et al documented that PDA in toy and miniature poodles is “a specific, localized, developmental anomaly which is genetically determined.” This mode of inheritance does not follow a simple Mendelian pattern. When normal dogs were crossed with dogs that had PDA, some offspring had PDA whereas others had an intermediate condition in which the DA closed only at the pulmonary arterial end to create a ductus diverticulum. This finding suggests that the trait is quasicontinuous, that is, a threshold trait with graded phenotypic expression. In other words, the chance of a dog having PDA and the severity of the abnormality increase with increasing amounts of defective genome from the parents. The results of these breeding experiments supported a two-threshold model of inheritance. When the first threshold is reached, a partial closure of the DA results in a ductus diverticulum. If the second threshold is reached, a PDA results. The lowest incidence of defective DA closure was noted in the offspring from normal dogs bred with dogs with apparent PDA (20%). An intermediate incidence was noted in the offspring when unaffected dogs with affected first-order relatives were mated with dogs that had ductus diverticula. The highest incidence (80%) of PDA was noted in offspring when both parents had PDA.

Buchanan and Patterson found similar structural abnormalities in sporadic cases of PDA in collies, cocker spaniels, German shepherds, Pomeranians, Shetland sheepdogs, and shih tzus, suggesting that there may be a similar genetic component in these breeds. A genetic basis for the occurrence of PDA has also been documented in Welsh corgis. This further supports the conclusion that dogs with a PDA should not be bred, even if the breed is atypical for the anomaly.

Presentation and Clinical Signs

PDA is overrepresented in female toy and miniature poodles; Maltese; Pomeranians; Shetland sheepdogs; cocker and English springer spaniels; keeshonden; bichons frises; Yorkshire terriers; and collies. When identified at a young age, dogs may have no clinical signs or may present with mild exercise intolerance and stunted growth. Owners may mention that they can feel the puppy’s heart “buzzing.” Many dogs with PDA are identified when they present for routine puppy vaccinations at 6 to 12 weeks of age, when a left-sided, continuous machinery murmur is auscultated. In most cases, a palpable thrill is noted.

Diagnostics

Thoracic auscultation is the primary diagnostic tool for detecting PDA. It is very important for veterinary clinicians to perform a complete thoracic evaluation when examining young dogs. Any heart murmur warrants further diagnostic evaluation.

The PDA murmur is typically so loud it can be auscultated all over the thoracic cavity, but the region of maximal intensity should be around the third intercostal space at the heart base. There are some exceptions to this characteristic murmur. A very large PDA may be auscultated as only a systolic murmur if pulmonary and systemic pressures are equalizing. Dogs with right-to-left shunts may have a faint diastolic murmur from pulmonic regurgitation, a split second heart sound, or no murmur at all. Other physical findings may include “water hammer” pulses, which are hyperkinetic, bounding pulses resulting from a large pressure gradient between the systolic and diastolic pressures. This occurs because of diastolic “run-off” through the PDA, causing a lower-than-normal diastolic pressure. A very small PDA may not have a palpable thrill.

Thoracic radiography is useful for evaluating the anatomic changes consistently seen in patients with PDA. Dorsoventral and lateral views of the thorax should be obtained for proper cardiopulmonary evaluation. The larger the PDA or the older the patient at the time of evaluation, the more prominent the radiographic changes will
be. Specific changes include progressive enlargement of the left atrium, left ventricle, aortic arch, and pulmonary arteries. The dorsoventral view is helpful for evaluation of the heart and great vessels. The cardiac silhouette may appear elongated due to aortic arch enlargement cranially and left ventricular enlargement that extends the silhouette caudally. The right apical lung lobe vessels are considered enlarged if they are greater in diameter than the smallest diameter of the fourth rib. Extreme cardiomegaly may shift the heart into the right hemithorax. The most characteristic sign of PDA is an aneurysmal bulge in the aorta at the level of the ductus. This "ductus bump" can be seen radiographically as a lateral deviation of the left lateral wall of the descending aorta at the level of the main pulmonary artery (FIGURE 4); however, this change is not always present. On the lateral view, overperfusion of the lungs can be appreciated, as well as left-sided heart enlargement indicated by dorsal elevation of the trachea and increased sternal contact (FIGURE 5).

Electrocardiography (ECG) should be conducted to evaluate heart chamber enlargement. Tall R waves (more than 2.5 mV) or wide P waves are typically noted on a lead II tracing. In patients with advanced heart disease from PDA, atrial fibrillation or ventricular ectopy may be noted. Atrial fibrillation is a late change and is associated with a grave prognosis. It is the result of an incompetent mitral valve that allows such significant backflow of blood that the left atrium is severely stretched.

Echocardiography may not always be required as part of the workup of a suspected PDA, but it may confirm the diagnosis and help to identify any other cardiac defects. It can also demonstrate changes in cardiac wall thickness and chamber size. To visualize the PDA, the right parasternal short-axis and left cranial window views are most useful. Identification of PDA often involves finding a high-velocity, turbulent flow pattern in the pulmonary artery as the blood exits the PDA. This is best detected using color-flow Doppler imaging. The degree of shunted blood flow is reflected in the magnitude of left atrial and ventricular eccentric hypertrophy. The left ventricular outflow ejection velocity is normally increased along with transaortic and transmitral flow velocities. Increased ejection volumes may result in flattening of the intraventricular septum. Identification of PDA with echocardiography depends on the skill of the individual performing the examination. Nuclear scintigraphy can also be used to quantify left-to-right and right-to-left shunts.

**Right-to-Left Shunts**

A small percentage of patients with PDA present with severe exercise intolerance and/or pelvic limb collapse during exercise. These patients have a reversal of normal flow through the PDA that can be documented with color-flow Doppler imaging. This reversal mixes nonoxygenated blood from the pulmonary artery with oxygenated blood from the aorta. The hallmark of a right-to-left shunt is differential cyanosis of the caudal half of the body. This is best visualized on examination of mucous membranes. Cyanosis can occur cranially as well.

Some dogs are born with a persistent right-to-left shunt, whereas others may develop right-to-left shunts over several weeks to months. Most dogs with right-to-left shunting have
such large PDAs (grade 6) that there is no resistance to blood flow. Normally, pulmonary vascular resistance is 20% of systemic vascular resistance after birth, so in dogs with grade 6 PDAs, the pulmonary circulation should receive five times the blood flow that the systemic circulation receives, causing left-sided heart volume overload that rapidly leads to left-sided heart failure, pulmonary edema, and death. However, because not all of these dogs die immediately after birth from left heart failure, it is theorized that pulmonary vascular resistance does not decrease to normal at birth, but rather remains persistently elevated in dogs with a congenital right-to-left shunt. This would provide resistance against potentially massive shunting and rapid death. However, this adaptation eventually results in life-threatening pulmonary hypertension. The pulmonary arteries retain vestigial medial hypertrophy, which slowly increases local vascular resistance. These arteries become damaged from persistent pressure, and growth factors become elevated, leading to smooth muscle cell hypertrophy/hyperplasia and connective tissue protein synthesis. The resultant medial hypertrophy and intimal proliferation lead to progressive narrowing and increasing pulmonary vascular resistance. In dogs with large PDAs, between 3 months and 3 years of age, pulmonary vascular resistance exceeds systemic vascular resistance and blood flow reverses, creating a right-to-left shunt. The distinctive PDA murmur is lost during this transition.

A right-to-left PDA becomes clinically important when it shunts a large amount of nonoxygenated blood from the pulmonary circulation (pulmonary artery) into the systemic circulation (aorta). This can reduce circulating arterial oxygen tension to a level typically between 30 and 45 mm Hg (normal: ~100 mm Hg). Because the subclavian artery and brachycephalic trunks branch off of the aorta cranial to the PDA, most of the mixing of nonoxygenated and oxygenated blood occurs in the descending aorta, resulting in inadequate oxygen delivery to the caudal portion of the body. Exercise exacerbates cyanosis by decreasing systemic vascular resistance. In the meantime, the body attempts to compensate for worsening oxygen deprivation and chronic renal anoxia by increasing erythropoiesis. The resulting polycythemia is a helpful adaptation when the hematocrit levels. Eventually, the blood becomes hyperviscous, slowing the circulation and further impairing oxygen delivery. The pulmonary vasculature responds through continued vasoconstriction, which perpetuates the pulmonary hypertension.

**Treatment and Outcome**

**Interventional Therapy**

PDA in dogs can be managed with medical therapy or occlusion using either open surgical ligation or minimally invasive techniques. In human pediatric medicine, nonsurgical occlusion of PDA is accomplished by using prostaglandin synthase inhibitors (e.g., indomethacin, ibuprofen) to stimulate natural closure. The use of prostaglandin synthase inhibitors does not appear to be effective when there is hypoplasia of the smooth muscle of the DA, however, which is the most common scenario in dogs. Moreover, dogs are generally diagnosed with PDA several weeks to months after birth, when the smooth muscle within the DA is no longer responsive to antiprostaglandin therapy. Due to the ineffectiveness of medical intervention, mechanical occlusion of the PDA remains the mainstay of treatment in dogs.

Since the 1950s, when the first surgical repair of canine PDA was performed, surgical ligation has proved to be an effective procedure in the proper candidates. The ideal canine patient for surgical closure of a left-to-right shunting PDA is a dog between 8 and 16 weeks of age with no concurrent cardiac disease and minimal secondary heart changes. Contraindications to occlusion of a PDA are right-to-left shunting, bidirectional shunting, or concurrent cardiac conditions that rely on the PDA for survival (e.g., tetralogy of Fallot). Older dogs with a recent diagnosis of a hemodynamically significant PDA should undergo surgery as soon as possible if they are reasonable surgical candidates. Severe secondary myocardial failure does not preclude surgery. If CHF is present, it should be treated aggressively before surgery. Animals with pulmonary hypertension can undergo surgical ligation of the PDA as long as systemic pressures remain higher than pulmonary pressures. Older dogs may be at greater risk of complications during and after surgical intervention.

**Preoperative Procedures**

Along with physical examination, a complete blood cell count and serum chemistry analysis should be part of the database for patients with PDA. Dogs with radiographic evidence of pulmonary edema should be treated with furosemide for 24 to 48 hours before surgery. In patients younger than 1 year or patients in very poor physical condition, preoperative fasting should be less than 6 hours in duration. A balanced electrolyte solution should be administered intravenously at a standard rate (e.g., 11 mL/kg/h) and may be supplemented with dextrose solution in very young puppies or dogs with documented hypoglycemia. A jugular catheter is useful for blood draws and rapid infusions of fluids or blood products and may be appropriate in high-risk surgical patients. Preoxygenation can be used before anesthesia induction. Patients should be premedicated with an opioid. I do not routinely premedicate these patients with anticholinergics, giving these drugs only if the heart rate drops below acceptable levels; however, routine premedication with anticholinergics has been advocated. Anesthesia is induced by IV injection of an ultrashort-acting barbiturate (e.g., thiopental, propofol) or inhalation of gas via face mask. Etopidate, a
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Carboxylated imidazole derivative, may be a more favorable induction agent for patients with CHF.

Surgical Ligation

The first surgical ligation of a PDA in a dog was recorded in 1952. Surgical interventional techniques have not varied significantly since 1967, when a detailed surgical report of a successful PDA ligation in a 4-week-old male mongrel puppy was published.21

The standard approach for PDA ligation in dogs remains a left fourth intercostal thoracotomy. The patient is positioned in right lateral recumbency, with a small rolled towel placed under the cranial thorax to maximize exposure by arcing the chest and spreading the ribs on the left side.22 The forelimbs may be secured in gentle extension. The patient’s entire thorax should be clipped and prepared just beyond the dorsoventral midlines, extending cranially to the point of the shoulder and caudally to the last rib. It is helpful to clip the caudal aspect of the proximal antebrachium, including the elbow, as it is often in the surgical field. The skin incision is centered by counting the intercostal spaces back from the palpable 12th space. A generous, curved skin incision is made from just ventral to the vertebral processes to ventral to the costochondral junction along the desired intercostal space. The incision is continued down through the subcutaneous tissue and cutaneous trunci muscle. The latissimus dorsi muscle is sharply incised along the same line, although some surgeons prefer to retract the latissimus muscle dorsally, which may decrease the postoperative discomfort associated with muscle transection but may also limit visualization.

Once deep to the latissimus dorsi, the surgeon should recount the intercostal spaces by palpating the first rib and counting caudally to the proposed incision site. The ventrally located scalenus muscle serves as a landmark for a fourth intercostal incision because the muscular portion of the scalenus inserts on the fifth rib, with fibrous bands extending the insertion to the eighth and ninth ribs. The scalenus muscle is incised over the fourth intercostal space, and the serratus ventralis muscle fibers are separated and retracted dorsally or sharply incised. The incision through the external and internal intercostal muscles is centered between the ribs to avoid the intercostal vessels running in the costal groove on the caudal aspect of the ribs. Metzenbaum scissors are used to lift the muscles away from the pleura and to minimize risk of iatrogenic injury to the underlying lungs.

Once the pleura is identified deep in the intercostal muscles, it should be carefully penetrated with scissors during exhalation. This creates a pneumothorax, allowing the lungs to fall away from the incision. The patient must be manually ventilated at this time. The pleural incision is carefully extended dorsally and ventrally. When the incision reaches the costochondral junction, care must be exercised to avoid injury to the internal thoracic artery where it courses subpleurally close to the sternum. Moistened laparotomy sponges or 4 × 4 gauze sponges (for smaller patients) are placed cranially and caudally along the exposed incision, providing protection from desiccation and cushioning the soft tissues from the Finochietto retractor. This retractor is inserted between the ribs and slowly opened until adequate exposure is achieved without injuring the ribs. After a cursory examination, the cranial lung lobe is carefully packed off caudally with a moistened 4 × 4 gauze sponge.
At this point, the surgeon should pause and identify the normal anatomy of the thoracic cavity, appreciating the orientation of the aorta and pulmonary artery. The phrenic nerve can also be seen where it courses ventral to the vagus across the width of the heart. The vagus nerve typically courses over the PDA, providing a good anatomic landmark. The location of the PDA can be verified by gentle palpation of the associated thrill (FIGURE 6). The vagus nerve is delicately isolated by sharp dissection of the surrounding tissues at the level of the ductus and gently retracted with umbilical tape, red rubber stays, or suture (FIGURE 7). The ductus is dissected bluntly without entering the pericardium. Using right-angle forceps or Lahey bile duct forceps, dissection is initiated parallel to the caudal aspect of the ductus. The tips of the forceps are inserted in a closed position and slowly opened to gently separate the tissues surrounding the PDA. The right-angle forceps are withdrawn with the jaws partially open. Closure of the forceps while in the tissues could result in inadvertently grabbing or tearing the fragile wall of the ductus. Once a lateral-to-medial dissection plane is opened on the caudal aspect of the ductus, a cranial dissection plane can be established. This dissection plane is created by angling the right-angle forceps approximately 45° and using the same technique of gentle dissection and withdrawing the forceps with the jaws open. Due to the difficult angle of cranial dissection, straight or curved forceps or hemostats are sometimes used.

After adequate dissection is completed cranial and caudal to the ductus, medial dissection is attempted. The right-angle forceps are carefully passed around the medial aspect of the ductus from caudal to cranial, and a tunnel for suture passage is created through gentle and persistent dissection. The final portion of the medial dissection can be frustrating, as there is frequently a thin curtain of tissue preventing the emergence of the forceps tips into the cranial dissection plane. Inexperienced surgeons are cautioned to exercise patience at this point and to continue the slow, steady dissection on the medial aspect. Some surgeons recommend using a moistened cotton-tip swab to aid in dissection. Utmost caution is necessary at this point because most episodes of hemorrhage caused by a ductal tear occur during the medial dissection of the ductus (FIGURE 8). When the tips of the right-angle forceps can be safely opened cranially, a strand of suture is introduced into the jaws for passage around the ductus. A pliable, heavy, nonabsorbable suture material (e.g., 1, 0, or 2/0 silk or Dacron) is recommended for ductus ligation (FIGURE 9). The ductus is closed by double ligation; the surgeon can either pass two separate strands of suture material or create and pass a loop of a single strand that is then cut to yield two pieces (FIGURE 10).

With either technique, the surgeon must be careful not to cross the suture strands on the medial aspect of the ductus. Also, the surgeon should never force the passage of the suture material around the ductus. If the suture does not pass smoothly, the forceps are opened, the suture is released, and then the forceps are withdrawn and replaced for another attempt at passage. Patience and adequate dissection around the medial aspect of the ductus will ultimately ease the passage of the suture material.

Once two strands of suture have been passed, they are checked to ensure that they are not entwined. The suture material should slide freely around the ductus but should not be aggressively manipulated, which can cause the suture to erode through the ductus wall, resulting in catastrophic hemorrhage. When the surgeon is ready to occlude the ductus, the sutures are tied. The suture closest to the aorta is ligated first. The ligature is slowly tightened, and the knot is secured with a minimum of five throws. The patient may develop a drop in heart rate at this time (i.e., Branham sign), a reflex bradycardia due to a sudden increase in aortic pressure as the PDA is ligated. Some authors recommend attenuating the ductus over a period of 2 to 3 minutes to minimize...
Once the loop of suture is transected, two strands are separated. One is retracted toward the aorta (upper strand) and the other is retracted toward the pulmonary artery (lower strand). The strand closer to the aorta is tied first.

Chest wall closure is begun using four to eight evenly spaced circumcostal sutures that are placed by skimming the cranial aspect of the fourth rib and taking a slightly larger bite of tissue around the caudal edge of the fifth rib. The type and size of suture material depend on surgeon preference. I (K. D. B.) use absorbable sutures (e.g., PDS II, Ethicon) in sizes ranging from 1 to 3/0, depending on patient size. Other surgeons may prefer using an appropriately sized nonabsorbable suture. An assistant (if available) crosses two adjacent sutures to appose the ribs and allow the surgeon to tie the initial suture. This process is repeated until the ribs are securely apposed. Alternatively, a rib approximator can be used to appose the ribs, taking care to avoid overlapping them. A technique of placing transcostal sutures (i.e., suture passed through holes drilled in the body of the rib) has been described to avoid entrapment of the intercostal nerve on the caudal surface of the rib in the circumcostal suture, which can cause postoperative pain.26 This report did not discuss the biomechanical effects of drilling multiple holes in the ribs. The serratus ventralis and scalenus muscles are then sutured back to achieve one layer of soft tissue closure. The incised edges of the latissimus dorsi muscle are reaposed or released from their retracted position.

Once an airtight seal is achieved, the thoracic cavity is evacuated via a butterfly catheter, needle, or chest tube with a three-way stopcock for easier aspiration and disposal of air and fluid. The panniculus, subcutaneous tissue, and skin are closed routinely. I (K. D. B.) prefer to place skin sutures in toy-breed puppies, but an intradermal suture pattern or skin staples can be used, although these may not engage well in smaller animals. With the patient still under anesthesia, a light wrap may be placed over the thoracic incision to avoid trauma to the incision and provide light support to the thorax. The wrap includes a nonadherent contact layer, an absorbent layer, and an outer protective layer. Great care must be taken to ensure the thoracic wrap does not impinge on the respiratory excursions of the patient and cause dyspnea, hypoxia, and cyanosis. An excessively tight thoracic bandage could result in the death of a small patient in the postoperative period. Most patients are discharged approximately 48 hours after PDA ligation surgery with a loose “t-shirt” or stockinette covering the thorax.

Variations in Surgical Technique

Several variations on dissection and ligation technique and suture passage exist. Because the medial aspect of the PDA is potentially weak and catastrophic hemorrhage is possible, sterile cotton swabs can be used for dissection of the cranial and caudal aspects of the PDA in lieu of forceps.25 As for variations on suture passage, one author recommends using right-angle forceps to pass a knotted double strand of suture with the loop cut off. This technique is meant to avoid engaging soft tissue from the blind side of the PDA.
by preventing complete closure of the jaws of the forceps, which are forced slightly apart by the knot. Another technique uses a stainless steel orthopedic wire loop (18- to 20-gauge) passed from caudal to cranial to safely pull suture around the medial aspect of the ductus.

Perhaps the second most commonly used suture passage technique is the Jackson-Henderson method. This technique is designed to avoid "tedious PDA dissection by drawing the ligatures from the dorsal and medial aspects of the aorta." The aorta is dissected free from its mediastinal pleura between the left subclavian artery and first intercostal artery to create a space for suture passage. Right-angle forceps are placed cranial to the ductus around the dorsomedial aspect of the aorta. The suture loop is accepted from this location and pulled through in a ventrolateral direction. Next, the two free suture ends are pulled through from a right-angle forceps that begins its passage caudal to the ductus and finishes at the dorsomedial aspect of the aorta (FIGURE 11). Once drawn ventral to the ductus, these strands are divided and tied as two individual ligatures. Jackson and Henderson reported no size limitation and no complications using this technique on 11 dogs. Since this description, there has been much debate as to whether this technique increases the risk of complications. Stanley et al reported increased short- and long-term complications using the Jackson-Henderson technique; in this study, 19% of the dogs that underwent PDA ligation via the Jackson-Henderson technique had intraoperative complications, including tearing of the thoracic duct, rupture of an aortic aneurysm, and tearing of the caudal aspect of the ductus. Using color-flow Doppler imaging, 53% of Jackson-Henderson ligations had residual flow compared with 21% for the traditional technique. Clinical outcomes of cases with residual flow were not stated. Stanley et al suggested that complete ligation is not achieved with the Jackson-Henderson technique due to excessive soft tissue inclusion in the ligatures. Bellenger et al thought that this technique decreased the risk of hemorrhage but increased the risk of residual shunting. Other studies showed no adverse effects of the Jackson-Henderson technique on patient outcome.

In 1971, Breznock et al reported on the use of tantalum hemostatic clips for the ligation of PDA to avoid medial dissection and facilitate application. One large (10-mm) tantalum clip was placed over the ductus and compressed in as little as 5 minutes with no intraoperative hemorrhage attributable to the ductus. These clips close from the tip toward the body, supposedly avoiding any extrusion of the PDA vessel. Tantalum clips are rarely used in humans due to the risk of recanalization. More recently, the use of tantalum hemoclips was reexamined in a study of 20 dogs, 19 of which had successful complete occlusion of the PDA. Medial dissection was avoided, but hemorrhage comparable to that associated with routine surgical ligation was noted at the cranial dissection site (10%). One dog had incomplete occlusion with persistent but minimal residual flow at day 560 postoperatively. This study did not report any recanalization and had a mean follow-up of 799 days.

### Surgical Complications

Overall, surgical complications are minimal for routine PDA ligation performed by an experienced surgeon. Mortality is reported at 0% to 2% for surgeons who have performed more than 100 such operations. Complications include hemorrhage, laryngeal dysfunction, air embolization, central nervous system hypoxia, myocardial hypoxia, hypothermia, and hypercapnea/hypocapnea with subsequent respiratory acidosis or alkalosis.

The most serious complication encountered is traumatic injury to the PDA. The occurrence of intraoperative hemorrhage was reported at 6.25% in a series of 64 cases. If hemorrhage occurs, mortality increases significantly, ranging from 42% to 100%. Ruptures generally occur intraoperatively, although there have been reports of postoperative deaths from rupture of an aortic aneurysm 5 hours to 16 days after corrective PDA surgery. Hemorrhage occurs most often during dissection around the medial aspect of the PDA near the right pulmonary arterial junction while the surgeon attempts to visualize the tips of the right-angle forceps. Hemorrhage is seen from the medial aspect as the forceps are withdrawn. At this point, the surgeon must decide whether to continue with the planned ligation or abort the attempt. Small ruptures typically respond to digital tamponade but may worsen with further dissection. In these cases, one option is to close and reoperate in the future, but the potential for adhesions makes a second attempt more
If the tear in the ductus is large and does not respond to tamponade, large vascular clamps can be placed and large, deep, biting mattress sutures placed in an attempt to occlude the ductus and control the hemorrhage. Another technique involves division of the PDA with oversewing of the ends. To do this, a large tangential vascular clamp (e.g., Satinsky clamp) is placed on the aortic side of the ductus and an angled 45° or 90° vascular clamp is placed on the pulmonary artery side. The ductus is transected, and each end is secured with a buttressed continuous mattress suture oversewn with a simple continuous pattern. This technique is also recommended by some authors for PDAs that are greater than 1 cm in diameter and/or for aorticopulmonary windows. Several authors have described techniques to induce hypotension for the management of intraoperative hemorrhage. Nitroprusside has been used to lower blood pressure to 45 to 60 mm Hg to slow or stop ductal hemorrhage. Its effects can be seen within 10 minutes of initiating the IV infusion. Nitroprusside and phentolamine have also been used before PDA dissection to lower pressures and decrease the severity of hemorrhage in the event of ductus trauma.

Key Points

- Patent ductus arteriosus is the result of asymmetrical distribution of ductus smooth muscle, preventing complete closure of the ductus arteriosus.
- The aortic aneurysmal dilation may not resolve after successful ligation or occlusion of a patent ductus arteriosus.
- Patent ductus arteriosus is a heritable condition, and affected patients should not be bred.
- In the hands of an experienced surgeon, surgical complications should be minimal.
- Depending on the amount of defective genome that is inherited, manifestations range from an asymptomatic ductus diverticulum to a clinically significant patent ductus arteriosus.

Risk Factors for Surgical Complications

Over the past 4 decades, several large studies have reported risk factors associated with short- and long-term complications resulting from surgical PDA ligation. In 1976, Eyster et al20 reviewed 100 cases of PDA and found a mortality of 8% for dogs that were surgically managed. The best survival rate was seen in dogs that (1) did not have ECG evidence of atrial fibrillation, (2) had no clinical signs of CHF, (3) were younger than 2 years, and (4) weighed less than 23 kg. Dogs presenting with ECG documentation of atrial fibrillation and mitral regurgitation had a mortality of 50% within 1 month of surgery, whereas dogs with mitral regurgitation alone had a 5% mortality. Only 34% of dogs that were treated medically survived beyond 1 year. Another large review of 201 dogs that underwent surgical ligation of a PDA found no correlation between long-term patient survival and age, body weight, level of surgical training, or surgical technique.21 This review reported that 7% of patients died intraoperatively, and an additional 4% died within 1 month of surgery. Intraoperative complications such as hemorrhage from the PDA negatively affected long-term survival. In 2005, another study reported that age, weight, lethargy, preoperative treatment with angiotensin-converting enzyme inhibitors, and right atrial dilation on radiography were all negatively associated with survival. In this study, 92% of dogs survived to 1 year and 87% survived to 2 years. No dogs died of heart-related disease beyond the second year after surgery. Forty-two percent of dogs in this study had mitral regurgitation alone with survival.21 Recently, Eyster20 reported that age and size are not factors in successful surgical treatment of PDA.

Minimally Invasive Techniques

Minimally invasive techniques for PDA occlusion have migrated into veterinary medicine as the fields of interventional radiology and cardiology and minimally invasive surgery have grown. Currently, minimally invasive procedures for PDA occlusion can be divided into intravascular techniques and thoracoscopic surgery. Intravascular procedures are described briefly here, but veterinarians interested...
in these techniques should undergo training with an experienced surgeon.

Intravascular techniques for PDA occlusion involve the use of either thrombogenic coils or intravascular occluding devices (duct occluders or vascular plugs). Thrombogenic or embolization coils (Cook Medical; Bloomington, IN) are composed of surgical-grade stainless steel wire with incorporated prothrombic synthetic threads. The vascular occluders (Amplatzer Vascular Plug, AGA Medical, North Plymouth, MN; Canine Duct Occluder, Infiniti Medical, Haverford, PA) use nitinol mesh to create a plug or disk shape that expands within the ductus lumen to close the PDA. The duct occluders may incorporate a polyester fabric to help close the defect and promote tissue growth. Vascular plugs are placed in the lumen of the PDA, where the multiple layers of nitinol mesh result in progressive thrombosis of the vessel.38

For these devices, intravascular access is normally established through a surgical cut-down or with the Seldinger technique. The femoral artery is usually used for arterial access, although percutaneous catheterization of the brachial artery has been reported.39

**Use of Thrombogenic Coils**

When a PDA is to be occluded using thrombogenic coils, angiography is performed before coil deployment to evaluate the shape of the ductus. Ideally, the ductus should have a distinct funnel shape (taper) to allow the coil to be lodged at the narrow end immediately before it empties into the main pulmonary artery. Thrombogenic coils should not be used in dogs with a nontapering (type III) ductus due to the risk of the coil being swept into the pulmonary vasculature.40,41 After angiography, a guide wire is advanced through the catheter into the PDA and then the pulmonary artery. Next, another catheter housing a thrombogenic coil is advanced over the guide wire until it is in place. The thrombogenic coil is deployed under fluoroscopic observation. The coil position is evaluated, and the thorax is auscultated for changes in the nature of the heart murmur. If the cardiac murmur is still present, a second coil is deployed. This process is repeated until the characteristic murmur is no longer present on auscultation. The average patient requires two to three coils for occlusion of the PDA, but as many as 10 coils have been used.42 For patients in which the PDA is too short (<5 mm) or fails to taper (type III), alternative techniques using another type of occluder or surgical ligation are recommended to achieve PDA occlusion.

After an adequate number of coils have been deployed, the catheter and sheaths are withdrawn and the artery is sutured or tied off or digital pressure is applied to the site for 5 to 10 minutes. Moderate to severe hematoma formation is one of the most commonly reported complications after interventional cardiology procedures.

A published comparison of “coiling” versus surgery listed the primary advantages of coiling as its minimally invasive nature and the potential for decreased patient discomfort (TABLE 1). However, the advantages and disadvantages of each technique can be debated, and expectations should be modified based on the experience of the operator.

Complications reported with thrombogenic coils include coil dislodgment, inaccurate coil deployment, lameness after arterial cut-down and occlusion, significant residual flow, severe hemorrhage at the site of the arterial cut-down, pulmonary artery embolization, partial aortic deployment, hemolysis, and implant infection.40,42 The combined rate of morbidity and mortality with coiling is less than that reported with surgical ligation (approximately 1% compared with 2% to 8%),40 which is directly related to the surgeon’s experience with PDA ligation.4 The importance of experience should also be stressed in successful thrombogenic coil placement.

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**TABLE 1** Comparison of Surgical Ligation and Thrombogenic Coil Placement for PDA Occlusion

<table>
<thead>
<tr>
<th></th>
<th>Coil Placement</th>
<th>Surgical Ligation</th>
<th>Advantage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Equipment costs</td>
<td>$500,000</td>
<td>$1000</td>
<td>Surgery</td>
</tr>
<tr>
<td>Supply inventory</td>
<td>$5000</td>
<td>$1000</td>
<td>Surgery</td>
</tr>
<tr>
<td>Single-use supplies</td>
<td>$500</td>
<td>$100</td>
<td>Surgery</td>
</tr>
<tr>
<td>Client charge (academic costs)</td>
<td>$2500–$3500</td>
<td>$2000–$2500</td>
<td>Neither</td>
</tr>
<tr>
<td>Procedure time</td>
<td>1–3 hr</td>
<td>1 hr</td>
<td>Surgery</td>
</tr>
<tr>
<td>Procedure personnel</td>
<td>3</td>
<td>2</td>
<td>Surgery</td>
</tr>
<tr>
<td>Animal size</td>
<td>Limited</td>
<td>Any</td>
<td>Surgery</td>
</tr>
<tr>
<td>PDA shape</td>
<td>Limited</td>
<td>Any</td>
<td>Surgery</td>
</tr>
<tr>
<td>Success rate</td>
<td>90%</td>
<td>98%</td>
<td>Surgery</td>
</tr>
<tr>
<td>Days in hospital</td>
<td>1–2</td>
<td>2–3 (depending on clinic)</td>
<td>Coils</td>
</tr>
<tr>
<td>Postoperative monitoring</td>
<td>Minimal</td>
<td>Moderate</td>
<td>Coils</td>
</tr>
<tr>
<td>Animal discomfort</td>
<td>Minimal</td>
<td>Significant</td>
<td>Coils</td>
</tr>
<tr>
<td>Mortality</td>
<td>&lt;1%</td>
<td>&lt;2% (experienced surgeon)–8%</td>
<td>Coils</td>
</tr>
</tbody>
</table>

Complete PDA occlusion may be obtained in only 60% of dogs at the time of coil placement, with complete occlusion occurring in about 85% of dogs in the 90 days after placement. In most dogs with reported residual shunting, the shunting was considered "clinically insignificant." However, roughly 5% of dogs receiving thrombogenic coils for PDA occlusion may require subsequent placement of additional coils to achieve complete cessation of flow. Thrombogenic coils have also been used to achieve complete occlusion when there is residual blood flow after surgical ligation. A recent review of surgical ligation versus coil placement for PDA occlusion found no significant differences in procedure length or patient mortality. Surgical ligation did have a significantly higher number of "major complications" compared with thrombogenic coil placement, but the unusual nature of the complications described casts doubt on the relevance of this conclusion. It appears that both thrombogenic coil placement and surgical ligation are acceptable options for PDA occlusion; many continue to assert that when morbidity, mortality, cost, necessary equipment, availability, and limitations dictated by patient size are compared, surgery is still preferable.

Use of a Duct Occluding Device

The Amplatz canine duct occluder (Infiniti Medical; Malibu, CA) is a self-expanding plug used to obstruct blood flow through a PDA. The Amplatz occluder has a flat disk that secures it in place and an expanding "waist" to occlude the PDA (FIGURE 12). The dense wire mesh of the occluder stops blood flow through the PDA.

Placement of an occluder begins with angiography to determine the size of the ductus and the pulmonary ostium. An appropriate occluder is chosen based on these measurements. A guide wire is passed through the angiography catheter and directed across the PDA into the pulmonary artery. The angiography catheter is removed, and the deployment catheter is advanced over the guide wire and across the ductus. The occluder is deployed with the distal disk opening in the pulmonary artery, and then retracted until the disk is in contact with the artery wall. Further deployment of the device fills the ostium of the PDA with the waist of the occluder. At this point, the occluder is checked for correct and secure positioning. If the operator is satisfied with the placement, the restraining cable is released and the deployment is complete. If the positioning needs to be improved, the device is reconstrained and repositioned. Correct measurement of the PDA ostium and accurate device sizing are essential for successful deployment of a duct occluding device. One review reported that interventional occlusion may be best accomplished when a variety of devices are available. This report recommended the use of detachable coils for small PDAs (<4 mm in diameter) and the use of the duct occluder for larger PDAs (>5 mm in diameter).

Thorascopic PDA Occlusion

Thorascopic PDA occlusion has also been reported in dogs. In five dogs, titanium vascular clips were placed to occlude the PDA. Fascia was cleared from the cranial and caudal aspects of the PDA, but dissection around the medial aspect of the PDA was not attempted. Minimal complications were encountered, and the dogs had rapid postsurgical recovery. The authors reported that the procedure was technically demanding but safe and effective. Accurate determination of the PDA diameter and vascular clip size was vital to ensure adequate occlusion. The report concluded that thorascopic PDA ligation was a viable alternative to surgical ligation in dogs weighing more than 7 kg with PDA diameters of less than 12 mm. Other important considerations for thorascopic procedures include equipment investment and level of expertise with minimally invasive surgery. Thorascopic procedures in veterinary medicine have included persistent right aortic arch resection, lung biopsy, pericardectomy, and thoracic duct ligation. These procedures are generally reported to have decreased postoperative pain, more rapid return to function, and fewer operative site complications. Experience with these procedures should enhance the potential for successful thorascopic PDA occlusion.

Long-Term Results

The recommendation for performing surgery as soon as possible has held through the years. Generally, ligation is considered curative if performed by 6 months of age. Early results suggest that this principle also applies to interventional catheterization and thorascopic techniques. There is a better chance that secondary mitral insufficiency and volume overload–induced myocardial failure will be reduced in younger patients. These changes may not be entirely reversible in older or larger dogs, but surgery is still indicated for left-to-right shunts in these patients. Follow-up radiography, auscultation, and echocardiography should be used to monitor patients postoperatively to document resolution of disease. The pulmonary vessels should be reduced to normal
size within 1 week, and the heart should return to normal by 3 months postoperatively. The aortic aneurysm will remain unchanged. This aneurysm is likely due to separation of the intraaortic segment of the ductus from the lumen of the aorta by a thin flap. Once the PDA is ligated, this region can still fill with blood and be visualized on radiography. If mitral regurgitation has resolved and the heart has returned to normal size, auscultation should also be normal.

Residual Flow

Failure to achieve complete occlusion and postoperative return of blood flow through a PDA are concerns in both human and veterinary medicine. In human medicine, residual flow in a PDA has potential long-term complications, including bacterial endarteritis and endocarditis of the main pulmonary artery and recanalization. In humans, recanalization can occur in less than 4 months in 6% to 23% of cases involving large PDA, the use of clips, or single or double ligation. Secondary bacterial endocarditis has been rarely reported in dogs, but perhaps of greater concern in veterinary medicine is the potential incidence of recanalization and the return of clinical signs of PDA. Recanalization has historically been cited in 2% to 3% of cases. Based on return of a machinery murmur and verification at surgery, Eyster et al documented the occurrence of recanalization to be 2%, with recanalization occurring twice in one dog. Lack of a murmur on auscultation does not rule out the presence of flow through a ductus vessel. One theory for recanalization is that ligation distorts the ductus, allowing the pulmonary artery and aorta to come into contact with each other. Friction from this contact could result in a new connection or fistula in an occluded ductus. Recanalization most frequently occurs cranial to the ligatures, further supporting this theory. Proponents of the hemoclip suggest that it does not cause distortion of the pulmonary artery–ductus–aorta orientation and thus minimizes risk of recanalization in dogs. Recanalization in humans after use of a hemoclip has been documented. Another theory is that recanalization results from incomplete occlusion of the PDA. With the increased use of color-flow Doppler imaging, residual flow may be detected in approximately 18% to 53% of cases. In a human study that used an Rashkind occluder device, the largest drop in residual shunting was noted from 1 day to 6 months postocclusion due to ongoing fibrosis. This study found surgical ligation to have significantly less association with residual flow than the use of an occlusive device. One author suggested that a small amount of residual flow will usually resolve by 3 months postoperatively due to the formation of scar tissue. Corti et al suspected spontaneous duct closure at day 560 in a case that had inadequate postoperative closure of the PDA using a hemoclip. Others disagree, stating that if residual flow is present at 1 month postligation, it is unlikely to resolve spontaneously. Most agree that division and oversewing minimizes the risk of recanalization, but this technique is time-consuming and potentially increases mortality. The long-term consequences of residual shunting are suspected by some to be minimal, but this topic has not been well studied in dogs to date.

Conclusion

PDA is a common condition with a variety of treatment options for occlusion. When the most appropriate option is selected and treatment is instituted early and skillfully, patients can have an excellent long-term prognosis.

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1. Dogs that are not treated surgically for a large PDA typically experience heart failure by what age?

a. 3 months  
b. 6 months  
c. 9 months  
d. 1 year

2. Dogs of which breed should not be bred if a PDA is documented?

a. toy poodle  
b. golden retriever  
c. keeshond  
d. all of the above

3. Color-flow Doppler imaging has shown a residual flow rate of ______ after occlusion.

a. less than 5%  
b. 5% to 12%  
c. 18% to 53%  
d. 25% to 28%

4. What is the incidence of PDA in dogs with congenital heart defects?

a. less than 5%  
b. 10% to 15%  
c. 25% to 30%  
d. 35% to 45%

5. A normal DA is primarily composed of ______ tissue.

a. elastic  
b. smooth muscle  
c. skeletal muscle  
d. collagen

6. What is the ideal conformation of a PDA for coiling procedures?

a. wide, nontapering  
b. funnel-shaped, tapering  
c. “window”  
d. all of the above are amenable to coiling

7. Which finding is not consistent with a right-to-left shunt in a dog with a PDA?

a. polycythemia  
b. caudal body cyanosis  
c. grade 5 to 6 left-sided murmur  
d. exercise intolerance

8. Which approach is used for ligation of a PDA?

a. left-sided fourth intercostal thoracotomy  
b. median sternotomy  
c. right-sided fourth intercostal thoracotomy  
d. left-sided seventh intercostal thoracotomy

9. When performing surgery to correct a PDA, life-threatening hemorrhage is most likely during

a. the initial approach to the intercostal artery  
b. penetration into the pericardium  
c. dissection of the medial aspect of the PDA  
d. penetration of a major vessel (e.g., pulmonary artery, aorta).

10. Which situation/complication during or after a coiling procedure does not require placement of an additional coil?

a. coil dislodgment  
b. delayed occlusion  
c. pulmonary artery embolization  
d. residual flow